Brown Discoloration on the Face

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CASE

A 54-year-old woman was visited with a history of asymptomatic gray-brown discoloration of the facial skin at our dermatology clinic. The lesions first appeared on her chin and then became progressively darker and extended to her nose and, to a lesser extent, to the periphery of her face over a period of five years. She mentioned that the lesions worsened with heat and sun exposure.

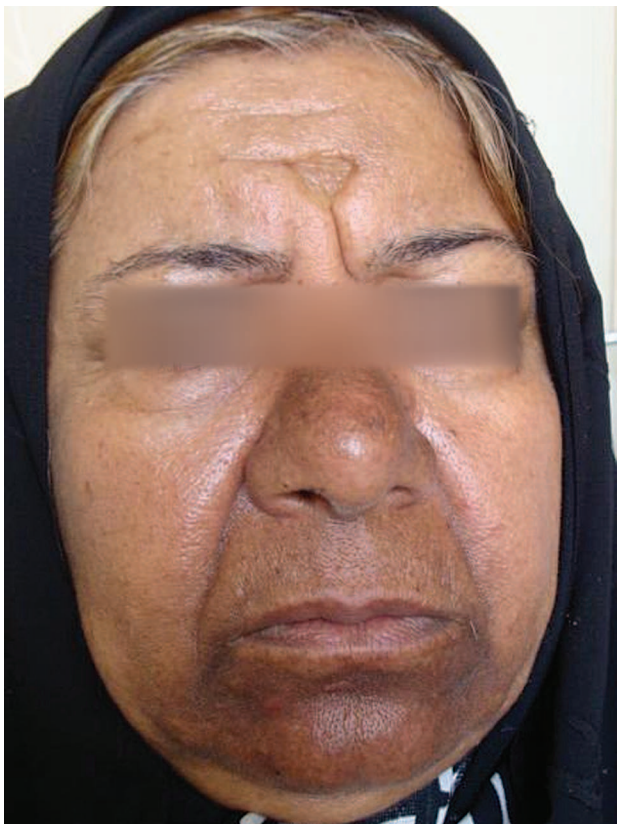
Her past medical history was unremarkable. The patient's medications included sunscreens, and hydroquinone lightening creams from many years ago.

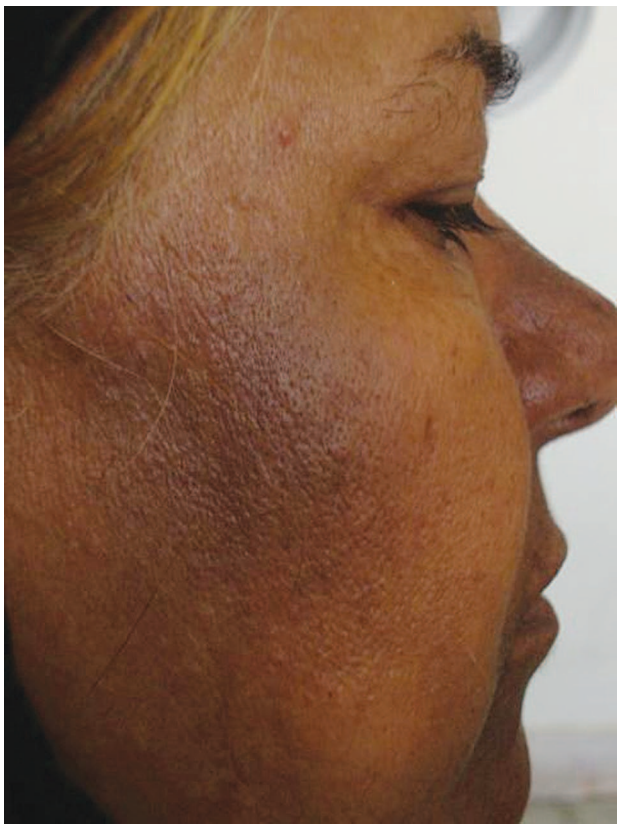
Physical examination revealed a previous depressed scar of cutaneous leishmaniasis on her forehead, brown to black hyperpigmented macules and patches, more in her midface area, especially the nose and chin, with superimposed small pigmented papules scattered on normal and hyperpigmented facial skin (Figures 1,2).

She had no discoloration on other body sites such as the neck, hands or trunk and there was no evidence of arthritis or joint pain.

The complete blood cell count, urine analysis and creatinin levels were normal.

What is your diagnosis?





Figures 1,2. Gray-brown discoloration of the face.

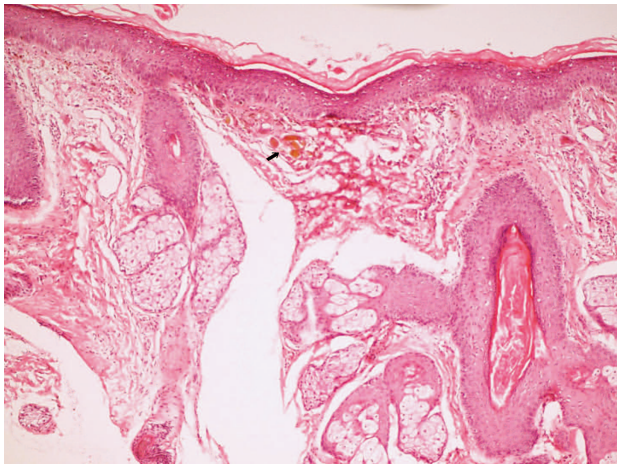


Figure 3. Round to oval ocher-colored deposits as well as solar elastosis, pigment incontinence and dilated vessels in the papillary dermis (H&E*10).

Microscopic findings

The histopathologic examination of her facial skin biopsy specimen showed mild spongiosis, pigment incontinence and multifocal round to oval shaped deposits, as well as solar elastosis and dilated vessels in the papillary dermis (Figure 3).

Diagnosis:

Exogenous ochronosis

Exogenous ochronosis is a paradoxical hyperpigmentation of the skin caused by long-term use of hydroquinone-containing bleaching creams [1] or topical application of phenol [2], resorcinol [3], or oral administration of antimalarials [4]. It commonly presents as asymptomatic blue-black macules on the malar areas, temples, inferior cheeks and neck [5].

This condition histologically resembles endogenous ochronosis in the skin but does not exhibit any systemic complications or the urinary abnormality [6]. It is clinically classified into 3 stages: stage I is characterized by erythema and macular sooty pigmentation, stage II by intense pigmentation and caviar like colloid milia, and stage III by papulonodular lesions [7].

The exact incidence of exogenous ochronosis is unknown [8]. It occurs almost exclusively in patients with a high skin phototype (Fitzpatrick's classification) [9].

There are various theories that explain the pathogenesis of exogenous ochronosis. The most accepted is that of Penneys' who attributed hyperpigmentation to the inhibition of the enzyme homogentistic oxidase by hydroquinone. This inhibition leads, like in endogenous ochronosis, to the accumulation of homogentistic acid that polymerizes to form ochre-colored pigmentation in the papillary dermis [10].

Histologic examination of exogenous ochronosis lesions reveals yellow-brown banana shaped fibers in the papillary dermis. Homogenization and swelling of the collagen bundles is noted and a moderate histiocytic infiltrate may also be present [6].

Exogenous ochronosis is largely refractory to topical agents, including tretinoin, cryotherapy, trichloroacetic acid, sunscreens, and corticosteroids. Clinical improvement has been reported after treatment with oral tetracycline, dermabrasion, and CO_2 laser irradiation; however, results are not uniform and the condition remains difficult to treat [11].

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